RUL 4-8-4

BIOLOGICAL HAZARDS FROM PROJECT RULISON

W. L. Robison and L. R. Anspaugh

Bio-Medical Division
Lawrence Radiation Laboratory
University of California
Livermore, California 94550

BIOLOGICAL HAZARDS FROM PROJECT RULISON

W. L. Robison and L. R. Anspaugh

Bio-Medical Division
Lawrence Radiation Laboratory
University of California
Livermore, California 94550

ABSTRACT

Project Rulison, an underground nuclear detonation designed for the stimulation of natural gas reservoirs, was detonated on 10 September 1969. When flaring of the natural gas is begun the major isotope to be released will be tritium (^{3}H).

Several questions have been addressed concerning the biology and physics of tritium. The general conclusions are that ordinary absorbed dose calculations based on energy uniformly distributed throughout a volume are sufficient to describe observed effects. No special mechanisms need to be considered in order to account for the degree of the effect observed. Also, organic molecules and especially deoxyribonucleic acid (DNA) have ^{3}H-^{1}H ratios essentially the same as that of the body water when animals are constantly exposed to ^{3}H. It appears there is no increase in the incorporation of tritium into DNA as a result of the ingestion of tritiated DNA by ascending trophic levels. The concentration of ^{3}H in the environment seems to be the controlling factor.

Dose estimates to the surrounding population from both inhalation and food chain pathways have also been made from source terms predicted for the Project Rulison operation. From these dose estimates risk calculations for genetic death, leukemia, cancer and non-specific life-shortening have been made for the 1000 persons within 15 miles of the Rulison site.

The total detriment to the first generation (genetic death plus non-specific life-shortening) if one uses the maximum estimated dose via the food chain of 30 mrem (a dose which could easily be reduced by employing appropriate precautions) is 2.6×10^{-2} lives lost per 1000 persons. In the interest of public safety these source terms, dose estimates and risk calculations have been made in a very conservative fashion such that upper limit risks are reported.

.026 / 100,000

PREFACE

The following analysis of the radiological hazards of Project Rulison was undertaken at the request of the Nevada Operations Office of the U.S. Atomic Energy Commission.

The meterological source terms for much of the analysis were supplied by the Environmental Sciences Service Administration, Air Resources Laboratory, Las Vegas. The total radionuclide inventory was supplied by the Los Alamos Scientific Laboratory.

The analysis which we have done is not complete in that we have not considered all possible routes of entry of radionuclides to man. Other groups have been requested to perform analyses in addition to those considered here. Questions relating to hydrology have been investigated by the U.S. Geological Survey and Isotopes, Inc. Hazards via some food chain routes not considered in the present analysis have been evaluated by the Battelle Memorial Institute, Columbus. Many additional evaluations have also been performed by the U.S. Public Health Service Southwestern Radiological Health Laboratory and the Nevada Operations Office.

It should be understood that the following analysis is therefore not a complete analysis of all radiological hazards, nor was it intended to be. Several specific questions are addressed in the following analysis; the absence of other considerations should not be construed as meaning that we felt them to be unimportant, but that they were being considered in detail by others.

I. Introduction

Project Rulison,[1] the second experiment in the stimulation of natural gas reservoirs by nuclear explosives, was detonated on 10 September 1969. It is presently planned[2,3] to re-enter the cavity six months postshot and to begin flow testing to determine the cavity volume produced by the explosive and the rate at which natural gas will flow from the "stimulated" reservoir. During the flow tests, the natural gas produced will be flared and consequently tritium in the form of water vapor (HTO) will be released to the environment.

The purpose of this paper is to examine the off-site radiological hazards associated with the Rulison re-entry and testing operations. Major attention will be devoted to tritium since it is considered to be the major potential source of radiation exposure. The validity of the maximum permissible concentration (MPC)[4] or radioactivity concentration guidance (RCG)[5,6] concept as it pertains to tritium will be discussed as will the possible dangers and pitfalls in applying the RCG values to the general public if possibilities exist that food sources will also be contaminated. Estimates will be derived for the maximum credible doses to the population at risk, and these will be used to estimate the maximum biological consequences that could ensue.

II. Problems Peculiar to the Biology and Physics of Tritium

Several factors have been discussed concerning the hazards of tritium relating both to its biology and decay characteristics. These

questions, which will be addressed below, may be summarized by the following: 1) Tritium (as tritiated water) is known to label organic molecules whereas the ICRP assumes it is distributed throughout the body water; should the MPC levels therefore be reduced? 2) Tritium can be incorporated into organic molecules where it turns over slowly compared to body water; might not models based solely on water turnover rates in aquatic ecosystems, for example, seriously underestimate the ultimate exposure to man who would ingest tritium labelled organic molecules? 3) Tritium decays by emission of a very low energy beta particle, and it has been suggested that ordinary dose calculations based on energy uniformly distributed throughout a volume may not adequately reflect the true damage observed, especially if tritium is incorporated into DNA and other nuclear components, and 4) Does tritium, if it is incorporated into deoxyribonucleic acid (DNA), also cause additional damage by transmutation of the DNA itself when the tritium decays?

The first question has been raised recently by Evans,[7] who analyzed the tritium content of lyophilization water and combustion water from seven tissues of 52 deer killed on the Savannah River Plant Site. His results indicated that the combustion water and free water contained the same tritium concentrations. He interpreted his results as meaning that the sustained exposure to tritiated body water would result at equilibrium in the labelling of all organic molecules equal to that of the

body water on a per gram hydrogen basis. He therefore concluded that the body burden due to chronic exposure to tritiated water would be 1.4 to 1.5 times higher than that predicted by the International Commission on Radiological Protection (ICRP) model.

Somewhat similar results have also been reported by Koranda et al.[8] They analyzed tritium content of body water and lyophilized organ residues of 95 kangaroo rats which had been living for generations in an elevated tritium environment surrounding the Sedan crater at the Nevada Test Site. They observed that the activity per gram of hydrogen in the lyophilized residue of six organs averaged 1.5 times higher than that found in the body water. They concluded after extrapolating their data to man in a situation of chronic exposure that the body burden and dose would be 1.8 times that predicted by the ICRP model.

These results, however, are in marked contradiction to those reported by Thompson and Ballou.[9] They exposed mature female rats to constant levels of tritium oxide, mated them after six weeks of exposure, kept the parent females on constant exposure during offspring development and nursing, and then maintained the offspring on the same exposure level for six months. At this time the offspring were removed from exposure to tritiated water, sacrificed at intervals, and the tritium content of combustion water from various tissues analyzed. For those animals sacrificed immediately, they observed that for most of the samples analyzed the combustion water contained 20 to 30% of the tritium

concentration in the body water during exposure. They interpreted this to mean that 20 to 30% of the organically bound hydrogen (both "freely exchangeable" as well as "firmly bound") was derived from body water.

If we accept this interpretation as correct, which seems reasonable from considerations of biochemical pathways of hydrogen incorporation,[10] we would have to assume that Evans' data are only interpretable if the deer were also consuming food containing tritiated organic molecules. This again seems reasonable since the exposure to tritium is chronic and would have provided adequate opportunity for the food sources utilized by the deer to be contaminated and the plant organic material to be labelled by tritium.

The kangaroo rat utilized in the studies of Koranda et al. is a unique animal which can subsist on dry seeds without the intake of any free water. This animal therefore derives a major fraction or all of its tritium from the ingestion of tritiated organics, and the tritium in its body water mainly from the catabolism of tritiated organics. These rats would exchange tritium between their body water and atmospheric water of lower tritium concentration and would also occasionally drink free water deposited by rain. Under these unique circumstances it seems reasonable that the body water tritium concentration should be lower than the combustion water tritium concentration.

We may conclude, then, that neither the study of Evans nor Koranda et al. may be interpreted as valid arguments for the reduction of the ICRP

recommended MPC's which are intended solely for either the breathing of air or the ingestion of water contaminated with tritium oxide. Clearly, the ICRP MPC values were not intended to apply to situations where food supplies are concomitantly contaminated.[4] There remains, however, the fact that some organically bound hydrogen is derived from body water, and that this organically bound reservoir of hydrogen atoms (or tritium atoms) has components that turn over slowly compared to body water.[8,9,11]

For purposes of evaluating chronic exposure to tritium, it might therefore appear appropriate to lower the MPC values by 10 to 20% to account for the additional body burden accumulated in this way. However, in comparison with the overall uncertainties involved in the establishment of MPC's which are given to only one significant figure, this would not appear to be useful or meaningful.

In situations involving acute exposure to tritium, somewhat different conclusions might be reached due to the long turnover times observed for the organically bound components. Even acute single administrations of tritiated water are effective in labelling the dry tissue solids of mice. One day following such an administration, Siri and Evers[12] observed that the tritium contained in dry tissue solids amounted to 7% of the injected dose with about 1.5% being firmly bound by metabolic processes. Some human data are also available concerning this problem. Snyder et al.[13] followed an individual accidentally exposed to tritiated water vapor and observed that the body water

elimination followed a curve described by the sum of two exponentials with half-lives of 8.7 and 34 days. The total tritium contained in the second component appears to have been something like 0.5% of that in the first component and contributed about 2% of the total dose. Reinig and Sanders[14] also studied a case of accidental inhalation where the dose, 46 mCi, was large enough so that the tritium excretion could be followed as long as 415 days. The observed urinary excretion pattern was assumed to indicate that the tritium was distributed among three compartments with half-lives of 6.14, 23.4, and 344 days. The highest percentages of assimilated tritium in the longer half-life compartments were 0.54% and 0.26%, respectively. Reinig and Sanders made conservative estimates of the dose contributions of the tritium in each of the compartments and concluded that the rapidly turning over compartment (body water) gave a maximum dose commitment of 3.52 rem whereas the slower compartments gave 1.37 rem. It therefore seems apparent that the dose calculated on the basis of distribution of tritium only within the body water should be increased by 40%. However, if we calculate the infinite dose using the ICRP model with a body water half-time of 12 days, the result is 5.8 rem.* This discrepancy is due to the conservatism

*Reinig and Sanders used a relative biological effectiveness (RBE) of 1.7 in their calculations. This value has subsequently been changed by the ICRP to 1.0,[15] but 1.7 was used in this calculation so it could be compared with the values of Reinig and Sanders.

incorporated into the ICRP model regarding the half-time of body water. Whereas the ICRP uses the value of 12 days, the two experiments mentioned above actually measured considerably shorter values, and in general the half-times measured by many experimenters average 9.5 days[16,17] rather than 12 days.

We would therefore conclude that the use of the ICRP model does not significantly underestimate the dose delivered by either chronic or acute exposures to tritiated water by ingestion or to tritiated water vapor by inhalation and skin absorption.

The second question relates specifically to the incorporation of tritium into DNA and other organic components of long turnover times. If such compounds become tritiated in low trophic levels of an ecosystem and are subsequently passed upward through a food chain, the possibility has been raised that the ultimate exposure to man eating such foodstuffs could be substantially higher than that predicted from the average concentration of tritium released to the environment.[18]

We might first ask the question whether tritium as tritiated oxide can be incorporated into DNA. Considerations of the biochemical pathways that generate nucleic acids clearly indicate that tritium contained in body water should be incorporated into DNA,[19] although there is a lack of experimental data on this point. Relevant data are those of Metzger et al.[20] They injected rats intraperitoneally with tritiated water

(25 μCi/g body weight), sacrificed the rats four hours later and then assayed the firmly bound tritium activity in macromolecular fractions of the liver. They observed approximately equal tritium concentrations (70 cpm/mg) in nucleic acids or proteins. The efficiency quoted for their tritium determination was 20%. Using this figure, we may calculate that the specific activity was 0.16 μCi/g. The concentration in the body water would have been about 40 μCi/g. Although the fractional activity is small, it is clear that even brief exposures to high concentrations of HTO are effective in incorporating trifium into rat liver DNA and proteins and that the levels of incorporation in the two classes of macromolecules are not essentially different. It also seems reasonable to assume that most biological organisms would have rather similar characteristics.

The question we would like next to address is to what extent the label in the DNA contained in an initial food link might be passed on to DNA's in subsequent links. Unfortunately, there appear to be no data available to make such analyses directly. While no studies are available on following tritiated nucleic acids through food chains, several studies have been done on the incorporation of tritiated DNA precursors into newly synthesized DNA.

Hinrichs et al.[21] injected tritiated thymidine (^{3}HTdR) intraperitoneally into mice and determined the percent of injected label that was incorporated into total body DNA. They observed that with

doses of less than 30 μCi per mouse, the amount of radioactivity incorporated into the total DNA of the mouse was less than 6% of the injected dose. Most of the injected activity was rapidly catabolized to HTO, as has also been observed in humans following intravenous injection of tritiated thymidine.[22]

Lambert and Clifton[23] compared the efficiency of incorporation of tritiated thymidine into rat DNA by the routes of intraperitoneal injection and ingestion. They observed that ingestion was only 1/8 as efficient in getting the tritium label into DNA. Combining the two experiments, we may conclude that following the oral ingestion of tritiated thymidine only 1% of the label is incorporated into DNA, and for mammals at least, we would expect little transference of tritium contained in ingested DNA into newly synthesized DNA.

It has been estimated that the weight ratio of DNA to body water is about 10^{-3} for both mouse[21] and man.[24] If similar ratios pertain to all plant and animal species, this would further argue that most of the tritium incorporated into newly synthesized DNA would come from sources and pathways other than the direct incorporation of tritium contained in ingested DNA. Additionally, there is no assurance that the ingestion of DNA precursors represents an adequate model for the ingestion of food containing intact DNA. Digestive processes may not provide precursors that can be readily utilized.

Relevant experimental data are those of Hatch et al.[25] who have studied the tritium activity contained in the liver DNA of the kangaroo rats taken from Sedan Crater. They observed that the tritium activity per g hydrogen was the same in the liver DNA as it was in lyophilized whole liver tissue. This indicates that these animals do not preferentially incorporate tritium into their DNA relative to whole liver tissue even though their tritium exposure is mainly derived from the ingestion of tritiated organic compounds.

For situations involving chronic exposure to relatively constant levels of tritium, we would conclude on the basis of the available evidence that there is no reason to believe that the tritium concentration in the long-lived macromolecular components of any trophic level organism would be significantly higher than that of other organic compounds or that of body water. (The kangaroo rat or any organism whose hydrogen intake is derived mainly from organic matter would be an exception to this as far as body water is concerned.) Situations involving acute exposure (or following the termination of chronic exposures) are clearly different, and the relative concentrations would be controlled by the relative turnover rates of the various organic and body water components. We have already discussed evidence indicating that following transient exposure, the long-lived organic components will eventually contain higher concentrations of tritium than the shorter lived components.

It should be kept in mind that these conclusions are based upon rather limited experimental data. However, the extrapolation of the available data to the Rulison situation can be made with reasonable confidence as the primary organisms of concern are mammalian, and the food chains are not complex. We do not feel nearly as confident about extrapolating these results to all members of complex (for example, aquatic) ecosystems involving a great diversity of organisms within food chains. Isotopic fractionation mechanisms for the hydrogen isotopes are known. Man, for example, exhales water vapor containing 94% of the tritium concentration in plasma,[26] and the pigeon has been reported to be unusually efficient in this process with the expired water vapor tritium level only about 50% of that of urine or blood.[12] Other fractionation effects observed between dueterium and protium have been summarized by Bowen.[27] How widespread these processes are is not known, and there appear to be no sufficiently detailed studies of the behavior of tritium in complex ecosystems so that the significance of these possible processes can be fully evaluated.

Question three, dealing with the dose estimates and subsequent effects as a result of the low energy tritium β particle, has been looked at in considerable detail by Bond and Feinendegen[28] and by Feinendegen.[11] They have considered their own work and the available literature. Some of the early somatic effects which have been observed after tritium exposure and compared with x-irradiation are cell killing in tissue

culture,[29-35] cell killing in intact mammals,[28] mitotic delay,[28,32] and various types of cytogenetic abnormalities.[36,37] Late term somatic effects which have been studied include carcinogenesis.[38] Genetic effects include both dominant lethal induction[39,40] and point mutations in the form of recessive lethals.[41-43]

Bond and Feinendegen[28] concluded that all of the short term somatic effects resulting from tritium exposure could be accounted for on the basis of the dose absorbed by the cell nucleus. The degree of the effect for any given absorbed dose as a result of tritium exposure was quantitatively similar to that observed following an equivalent dose of x-irradiation. In other words, no special calculations need to be considered other than the absorbed dose due to the random disintegration of ^{3}H atoms, regardless of whether the ^{3}H atom is in the form of HTO or actually incorporated in DNA as ^{3}HTdR. The same quantitative correlation between absorbed tritium dose and x-irradiation also appears to occur for long term somatic effects and genetic effects. In some cases the absorbed dose from ^{3}HTdR or HTO appears to be slightly less effective than an equivalent dose from x-irradiation while in other cases it appears to be slightly more effective. However, within the experimental error and technical difficulties in such experiments, it appears that the dose absorbed by the cell nucleus as a result of tritium decay quantitatively accounts for the degree of the effect and it does not exceed that expected from a similar dose of x-rays.

Lambert[34] has recently published results of experiments (not included in Bond and Feinendegen's analysis) designed to look at the death of intermediate and type B spermatogonia after irradiation of mouse testes. The testes were irradiated externally with x-rays or internally with ^{3}HTdR or HTO. Again, the quantitative effects considering the absorbed dose from tritium and that from x-irradiation were quite similar. The tritium irradiations were slightly more effective than x-rays in these experiments and HTO more effective than ^{3}HTdR. These results are similar to those of Johnson and Cronkite[35] where ^{3}HTdR and ^{60}Co γ rays were used for induction of spermatogonial killing.

Various experiments have been conducted to compare the effectiveness of tritium β irradiation for producing a given biological effect to the effectiveness of ^{60}Co γ irradiation or x-irradiation. In some experiments the tritium β seems to be slightly less effective for an equivalent dose while in others it seems to be slightly more effective. In some experiments where RBE values of 1.6 to 2.0 are calculated, the uncertainties in the dose calculations make the results very tenuous and the results should be regarded with circumspection.

Bond and Feinendegen[28] have pointed out that RBE's of 1.3 or 1.4 for tritium β irradiation relative to ^{60}Co γ irradiation are more like unity when compared to 250 kvp x-rays. Also, they have pointed out that RBE's of 1.3 to 1.7 which have been observed for a variety of endpoints

in intact mammals are based on calculations which assume uniform distribution in the body water and uniform whole body dose. However, a large part of an animal is bone and this component has a minimal uptake of HTO. When one takes into account this inhomogeneity in the distribution of tritium then the RBE value is nearly unity.

Recently the ICRP[15] has adopted a quality factor (or RBE) of 1.0 rather than 1.7 for beta energies less than 0.03 MeV; therefore, the quality factor for tritium has been adopted as 1.0 and the following dose calculations in this paper will be made with this value.

Question four concerns the possible effects of transmutation of DNA as a result of ^{3}H decay. Again, Bond and Feinendegen[28] have considered this problem. The general conclusion is that no experiments have shown that transmutation effects have any role in cell lethality (where results can be accounted for entirely by the absorbed doses) or other cell damage such as cytogenetic abnormalities where there is no correlation between the site of the chromosome break and the site of incorporation of the label.[37] Rachmeler and Pardee[43] have, however, apparently shown a transmutation effect in the induction of mutants in bacteria after labelling with $^{3}HTdR$. Bond and Feinendegen felt that transmutation could be invoked to explain the data of Kaplan et al.[41] who looked for sex-linked recessive lethals in Drosophila after labelling with $^{3}HTdR$. The distribution of lethal mutations along the x-chromosome was non-random and it was different from that observed

after x-ray exposure. However, in a more recent experiment Kaplan et al.[42] have looked at the distribution of mutations along the x-chromosomes after labelling with tritiated deoxycytidine (^{3}HCdR). The distribution differs slightly from that observed with ^{3}HTdR and also differs from that observed with x-rays. When the distribution resulting from the two different labels (i.e., ^{3}HTdR and ^{3}HCdR) were added together the results were like those observed with x-rays. This was interpreted as suggesting that the combined distribution is indicative of the regional content of DNA along the x-chromosome and that the difference in the distribution of lethal mutations produced by ^{3}HTdR and ^{3}HCdR demonstrates that variations in thymine and cytosine content may exist within the chromosomal DNA. Therefore, invoking transmutation effects is really not necessary in order to explain these results.

For the case of exposure to tritiated water, rather than ^{3}HTdR, which results in randomly labelled organic molecules the effect of transmutation would be negligible since tritium would not be preferentially incorporated into DNA and since DNA comprises only about 10^{-3} of the organic body weight.

There is one additional problem that should be considered and that is the unique situation of the fetus that is continually exposed to tritium throughout fetal life. The experiments of Khan and Wilson[44] and Thompson and Ballou[9] indicate that fetal exposure of rats leads to

the incorporation of tritium into organic molecules throughout organogenesis with subsequent long half-times depending on the site of incorporation. Also, several experiments have suggested, as stated by the ICRP,[45] that the effect per rad on the induction of neoplasia following fetal irradiation is greater than the effect on children and adults by a factor of between 2 and 10. Miller[46] has recently questioned the validity of such a conclusion, but has not presented any compelling evidence that it is incorrect.

These considerations would suggest that the critical segment of the population at risk will be the fetus. If the fetus is indeed ten times as sensitive as adults one could argue that dose standards should be reduced by an order of magnitude when applied to the exposure of pregnant women. The effect of tritium incorporation into organic molecules is much more difficult to assess in that it is not certain what percentage of the tritium under such conditions is bound to the various types of organic molecules. Obviously, some are much more critical than others.

III. Relevance of the RCG Values for the Exposure of the General Public

It seems appropriate before considering the particulars of Project Rulison to review briefly the nature of the RCG values,[5,6] and to point out how they may be completely inadequate when applied to the general public.

Historically, the RCG values are related to the maximum permissible concentration values (MPC) of the ICRP[4] which were calculated for the exposure of occupational workers. The primary dose standard had been set at 5 rem/year for the whole body and certain other tissues and the MPC's for air and water were calculated assuming that each route and each radionuclide was the only exposure the individual had to radioactivity.

A dose of 0.17 rem/year has been established for exposure of the general public, and therefore the RCG values for exposure of the general public were also lowered to 1/30 of that allowed for the continuous exposure of occupational workers. However, the key point is that the MPC's and the RCG's assume that there is no other route of entry of a radionuclide into that individual; this is a very reasonable assumption for occupational workers, but it certainly is not for exposure of the general public. As both the ICRP and the FRC have pointed out, one has to be very careful in applying the MPC's to any situation other than that for which they were calculated and corrections do have to be made when there is exposure via other routes. Many exposures of the general public as a result of nuclear activities also involve the very real possibility of the contamination of food chains by the radioactivity released. Clearly, the RCG's do not allow for this possibility, and we have already discussed in considerable detail how the concomitant contamination of food may influence the body burden of tritium.

As the ICRP and the FRC have stated, in order to be certain that the primary standard of 0.17 rem per year is not exceeded, it is necessary to consider all possible routes of entry of a radionuclide into man, not just what man may receive by breathing contaminated air or drinking contaminated water. As we will subsequently demonstrate for Project Rulison, the lack of such considerations could indeed lead to doses above the primary standard even though the air concentration values do not exceed RCG values.

IV. Estimated Doses from Tritium

In the Rulison basin the wind direction varies from daytime to nightime. Nightime flow is in a northerly direction down the valley while during the day the flow is up the valley in the opposite direction. In the daytime the flow will finally reach sufficient height to move with the prevailing northeasterly winds. Therefore, data have been presented for predicted air concentrations and ground depositions for a twelve hour daytime period and a twelve hour hightime period for both an accident situation and a normal flaring operation.[2]

The accident situation assumes that there would be no control over the gas flow at the wellhead and that uncontrolled blowout occurs through an open drill hole. The source terms for the accident case are calculated assuming that under such conditions 100% of the gaseous radionuclides present at 180 days post detonation would be released in a 24 hour period.

The normal flaring operation consists of controlled venting of the cavity volume. Source terms for tritium for the flaring case were developed assuming 20% of the cavity ^{3}H activity is vented in the first three-day flaring operation.[2,3] The predicted air concentrations and ground depositions from which the doses are calculated assume that the cloud travels in a straight line -- that is, there is no meandering of the plume. Actually, one would expect the cloud to meander during a twelve hour period. Therefore, because of the different wind directions during daytime and nightime and the probable meandering of the plume one would not expect any one person to be in the cloud for a 24 hour period and summing the dose over an entire day (or over a three-day flaring operation), would certainly produce a maximum dose estimate. Releases after the first three-day flare will involve smaller quantities of activity and they will be spread over many months. Therefore, such variables as plume meandering and wind direction will have a much greater effect and the dose received will be much less than that from the initial flare. The exposure estimates for the first three-day flare have been determined in a very conservative fashion and are probably reasonable estimates of the total dose one might possibly receive from the total flaring operation.

Knox[47] has supplied source terms for self-induced rainout which is a phenomena which may be observed at or near the wellhead. It consists of the condensation of water vapor out of a supersaturated gas cloud as it leaves the flaring stack.

Dose calculations will be made for the accident case and the normal flaring operation considering both air inhalation and ingestion via the food chain. The approach of Ng et al.[48] for determining unit rad depositions (the unit rad deposition values are the amounts of radionuclides in $\mu Ci/m^2$ that will result in a 30 year dose of one rad) was used for the dose estimates via the forage-cow-milk pathway. The unit rad deposition value for tritium via the forage-cow-milk pathway is 99 $\mu Ci/m^2$.

The unit rad deposition for the soil-root pathway has been calculated using the technique of Ng et al.[48] However, because of recent information concerning tritium movement in various ecosystems, we have made a few numerical changes in some of the constant parameters in the basic equations.[49] The approach used by Ng et al. assumes that tritium stays within the top 20 cm of the soil surface after deposition. Koranda[50] has recently shown, however, that the peak concentration of tritium moves up and down in the soil and reaches depths up to seven or eight feet. We have assumed, therefore, that on the average tritium is distributed in the top 100 cm of soil.

It was also assumed in the original analysis that the half-residence time for tritium in the soil was equal to its radioactive half-life of 12.3 years. However, recent evidence has shown the half-residence time for tritium to be about 29 days[51] in a tropical rain forest and about 18 months[50] in the dry desert region of the Nevada Test Site. Most areas

would probably fall somewhere between these two ecosystem extremes. In our analysis we used the longer of the observed half-residence times, i.e., 18 months.

With these two major changes in the equations used by Ng et al. the unit rad deposition for ^{3}H is found to be 1574 $\mu Ci/m^2$. Therefore, the dose received via the soil-root pathway would be at least 15 times less than that received via the forage-cow-milk pathway.

It might be mentioned here that the time of year when the flaring operation is conducted will have a considerable effect on the possible exposure via the forage-cow-milk pathway. If there is still considerable snow on the ground the cows will not be on pasture and the amount of tritium which would remain on the plants would be very much less than that which would be deposited on plants if the flaring were conducted after the snow melt. Also, the final tritium concentration in soil water would be less if flaring were conducted while there was still snow cover on the ground.

Another possible source of contamination that we have not considered is the contamination of water supplies. However, due to the large dilution factors involved and the ability to easily monitor such supplies in the Rulison vicinity, the dose from this source should be negligible. An evaluation of this problem has been published.

A. Accident Case

1. Inhalation

The first calculation concerns air inhalation at a distance of 5 km (distance from wellhead of nearest population) as a result of a daytime accident. At an effective release height of 300 m, which gives the highest concentrations, the predicted tritium air concentration is 3.5×10^{-8} Ci/m^3. If one uses the ICRP value for the volume of air breathed by standard man, i.e., 2×10^7 cm^3 per day or 10^7 cm^3 per 12 hours, then the numer of microcuries inhaled in a 12 hour period is 0.35 μCi. The amount inhaled is essentially all absorbed.[52] For a 70 kg man this results in a first year dose* of 3×10^{-2} mrem assuming a 12 day half-life for tritium in the body and a relative biological effectiveness (RBE) of 1.0.[15] The nightime air concentration at 5 km as a result of an accident situation is approximately a factor of 1000 below the nightime case and would, therefore, give a dose of 3×10^{-5} mrem for a 12 hour release period. At 15 km the nightime release would lead to a dose of 2×10^{-3} mrem while the daytime release would lead to a dose of 7×10^{-3} mrem.

2. Dry Deposition

The amount of tritium deposited on the ground at 5 km as a result of an accident is given as 1.5×10^{-5} Ci/m^2 for the daytime case. The

---

* First year dose and 30 year dose are the same for an acute exposure to tritium.

unit-rad deposition value for the forage-cow-milk pathway for tritium according to Ng et al[48] is 99 $\frac{\mu Ci}{m^2}$. This means a child drinking one liter of milk per day from cows grazing on pasture contaminated with ^3H at 100 $\mu Ci/m^2$ would receive a dose of one rem. The daytime deposition at 5 km therefore results in a dose of about 0.2 rem. The nightime deposition at 5 km is a factor of 1000 lower than this. If the entire chimney were emptied in a 24 hour period in the same location these two values would have to be summed. At 15 km the nightime and daytime depositions would lead to doses of 10 mrem and 40 mrem, respectively. As was pointed out previously, the unit rad deposition via the soil-root pathway is more than 15 times higher than that via the forage-cow-milk pathway and therefore the dose received via the soil-root system would be at least 15 times less than the doses calculated for the forage-cow-milk pathway.

B. Flaring Operation

1. Inhalation

For the flaring operation the initial calculation for the dose received as a result of air inhalation will be made for the daytime situation at 5 km. This is the maximum value predicted for air concentrations as a result of flaring and is, therefore, the limiting case for the air inhalation situation. The value for the predicted air concentration is 2.6×10^{-9} Ci/m^3. Again assuming an inhaled air volume for a 12 hour period of 10^7 cm^3 the short-term body burden is 0.026 μCi. This is a

factor of 100 less than the accident case or a dose of 3×10^{-3} mrem. The nightime release case at 5 km is a factor of 500 below this value. At 15 km the 12 hour daytime flaring would lead to a dose of 8×10^{-4} mrem, and the 12 hour nightime flaring would give an inhalation dose of 3×10^{-4} mrem.

The total inhaled dose at 5 km as a result of a three-day flaring period is then approximately 10^{-2} mrem. At 15 km the total inhaled dose would be about a factor of three lower than at 5 km or 3×10^{-3} mrem.

2. Dry Deposition

The deposition of tritium onto plants as a result of the flaring operation can be calculated using the source term value of 10^{-4} Ci-sec/m^3 for the integrated activity at 5 km for daytime release, and a value of 10^{-2} m/sec for the deposition velocity.[2] The result is 10^{-6} Ci/m^2. This is a factor of 100 less than the maximum accident case and gives a dose of 10 mrem for the forage-cow-milk pathway. Nightime deposition is a factor of 1000 lower and therefore leads to a dose of 10^{-5} rem. At 15 km the deposition from daytime release leads to a dose of 3 mrem while nightime deposition leads to a dose of 1 mrem. The doses resulting from the soil-root pathway would again be a factor of 15 lower than the above estimates for the forage-cow-milk pathway.

C. Rainout

Tritium may also be deposited on the ground surface by the process of natural rain bringing down the activity contained in the plume.

For the accident case, it has been estimated that the specific activity of this water might be between 1.8×10^{-3} and 4.8×10^{-3} μCi/g,[2] which can be compared to a RCG value of 1×10^{-3} μCi/g for drinking water. It would take a rain of about 2 cm to bring down enough activity to equal the unit rad deposition for the forage-cow-milk pathway. This amount of water, however, could not possibly be retained by the plant surfaces, and would move into the soil water. Dose contributions would therefore be reduced and would be less than the calculated dose arising from dry deposition and entry into the forage-cow-milk pathway.

The same conclusions would also apply to the flaring operation.

D. Self-Induced Rainout

Knox[47] has made an estimate for self-induced rainout at a distance of 2 km. Self-induced rainout is the condensation of water out of a super-saturated gas at or near the release point. His value of $60 \frac{g}{m^2\text{-hr}}$ for a 24 hour period gives 1440 g/m^2. The activity of the water is 10^{-7} Ci/g or, therefore, 1.44×10^{-4} Ci/m. If 25% of this self-induced rainout remains on the plants then there are essentially 3.6×10^{-5} Ci/m^2 which will lead to a dose through the forage-cow-milk pathway of 0.36 rem. Knox estimates the accident case would be some five times worse or approximately a dose of 1.8 rem. However, there are no people and no dairy cattle within 2 km of the wellhead.[2]

The results for the various situations are summarized in Table I. The significant point to be made from these data is that the food pathway

could contribute by far the greatest percentage of the dose. The dose contributed by air inhalation over a 12 hour period is trivial compared to the dose which could be received via the forage-cow-milk pathway as a result of such air concentrations. Therefore, simply keeping the air concentrations at or slightly below MPC is not sufficient. The daytime accident release is an example. The air concentration at 5 km for a 12 hour period is predicted to be 3.5×10^{-8} $\mu Ci/cm^3$ or a factor of two below RCG. However, even for this short period of time the resulting deposition of tritium on forage which could be incorporated in the food chain via the forage-cow-milk leads to a predicted dose of 0.15 rem which is very near the primary standard. The primary standard of 0.17 rem is the critical and controlling guide, and, therefore, the contribution of dose from all sources must be considered -- especially that via the food chain. Others[2] have considered the potential tritium exposure via other food chain pathways.

V. Hazards from other Radionuclides

A. Gaseous Activities

In addition to tritium, other radionuclides are present in the chimney in a gaseous form and are expected to be released during the flaring operation or in the event of an accident. The radionuclides of possible concern are ^{14}C, ^{37}Ar, ^{39}Ar, ^{85}Kr, and ^{133}Xe. Total inventory amounts and the predicted air activities are available.[2]

For the gases, the ICRP model considers hazards arising from submersion in a cloud of the gas. Calculations using this model indicate that the only gaseous radionuclide of significance is ^{85}Kr, which would contribute a dose equal to 25% of the dose due to inhalation of tritium. ^{14}C may also be inhaled, but would not contribute any significant dose by this route due to the relatively very small amount of this radionuclide present within the chimney.

Dry deposition values have also been given for ^{14}C.[2] Dose contributions from this radionuclide via the forage-cow-milk and the soil-root pathway have been evaluated using the model of Ng et al. and would be orders of magnitude lower than the dose contribution from tritium.

Ng et al.[48] have published unit rad deposition values for the noble gases for both the forage-cow-milk and the soil-root pathway. However, these values cannot be used in a meaningful fashion since deposition velocities for noble gases are not known. As an alternate approach, we have started with the maximum air concentrations given for the noble gases,[2] and assumed that the only way the gases can be deposited is to be absorbed by water. The maximum concentration of the noble gases in water was calculated using Henry's Law, and by assuming instantaneous equilibrium. This concentration of noble gases in water was then assumed to be directly transferred to man, and eliminated only by radioactive decay. These extremely conservative

assumptions indicated that ^{85}Kr was the noble gas radionuclide of most concern, but that the dose calculated in this manner was 10 times smaller than that calculated from the ICRP model of submersion in the original air concentration of ^{85}Kr. Therefore, any possible dose contribution by radionuclides of noble gases via food chain pathways is insignificant.

B. Particulate Activities

The hazards to man via the forage-cow-milk and the soil-root pathways for all particulate radionuclides contained in the chimney may also be estimated using the conservative values of Ng et al.[48] and compared to the tritium hazard.

This analysis may be made by considering the total chimney inventory at six months post detonation and the critical organ unit rad deposition values. The more conservative values for the infant have been used. Dividing the inventory (in μCi) by the unit rad deposition value gives the m^2-rad value which when multiplied by the fractional deposition per m^2 gives the dose in rads. More importantly, the m^2-rad value allows one to make a direct comparison of relative risk for different radionuclides.

This has been done using inventory values given in reference 2 and the results indicate that the radionuclides of most concern are ^{90}Sr, ^{106}Ru, ^{95}Nb, ^{89}Sr, and ^{137}Cs. For the most hazardous radionuclide, ^{90}Sr, the statement can be made that if its fractional deposition (or its air concentration) is 10^{-4} of that of tritium, it would

constitute a hazard equal to that of tritium. This same estimate of relative hazard for ^{90}Sr may also be derived by considering only the inventory relative to the RCG values for air concentration. However, essentially none of these isotopes are expected to exit the cavity.[2]

VI. Risk Estimates

There are approximately 1000 persons within a 15 mile radius of the Rulison site.[2,3] Since this is the critical population which could receive some exposure, calculations for the increased incidence of leukemia, cancer, life-shortening and genetic effects have been made on the basis of 1000 persons.

We will use dose estimates derived from the source terms for the normal flaring operation since it is the most likely situation to occur. The largest estimated dose to the population results via the food chain pathways. However, this is a controllable situation such that any dose via these routes (forage-cow-milk and soil-root) could be considerably reduced by appropriate Public Health measures. With this in mind, we will present risk estimates for doses received through both the forage-cow-milk pathway and through air inhalation. The largest dose via the forage-cow-milk pathway due to dry deposition is 30 mrem at 5 km while the largest total dose due to inhalation during a three-day flaring operation is 0.01 mrem at 5 km. Therefore, we will use these two numbers in our calculations.

It is assumed that the average absorbed dose resulting from tritium exposure is sufficient for making such calculations and the details of such calculations are given in the Appendix. The results are summarized in Table II. These are *conservative* upper limit estimates which are based on extrapolation from high doses and high dose rates. No account has been made of possible dose rate effects or possible repair mechanisms at these lower doses. Various studies indicate that such processes might be functioning in mammalian systems. For instance, radiation-induced mutation frequencies show a considerable dose rate effect with acute exposure resulting in approximately four times as many mutations as chronic exposure.[53, 54] The survival of irradiated cells, both in vivo and in vitro, has been shown to increase when the dose is delivered in a chronic or fractional manner rather than an acute manner.[33, 55] Also, fewer chromosome abberations are observed after chronic or fractionated exposure than after acute exposure.[56, 57] Chronic exposure studies on cancer induction in humans, however, are non-existent.

Repair processes have also been demonstrated in various organisms and cell lines after UV irradiation.[58-60] Unscheduled DNA synthesis in mammalian cells has been reported at x-ray doses of 5000 r[59, 61] and a type of "repair replication" after very large doses of x-irradiation has been observed in Hela-S-3 cells.[62] However, the relationship of this repair replication to cell recovery is still speculative. It has also been established that rejoining of x-ray induced breaks in the DNA of

mammalian leukemic cells occurs.[63] The rejoining process seems to be rather radio-resistant but again its relationship to cell survival has yet to be clearly defined.

Enzymatic rejoining of single-strand breaks in the DNA of bacteria has been reported by two different groups[64,65] and also the excision of thymine dimers and mismatched sequences by DNA polymerase has been observed by Kelly et al.[66] It appears the enzyme might be able to carry out both excision and polymerization indicating repair of damaged DNA. The relationship of all the observed repair mechanisms to cell killing, or more importantly, to the induction of various types of cancer and genetic effects, is a major question in radiation biology.

It is possible, therefore, that dose-rate effects and repair processes might, for low dose exposure, reduce the damage below that which is predicted by linear extrapolation from high doses and high dose rates. However, in order to be conservative in these calculations we will proceed on a linear extrapolation hypothesis from the observed effects at higher doses.

When the Appendix was originally written,[67] we felt that the weakest evaluation concerned the estimation of the magnitude of non-specific life-shortening, and a very conservative estimate of 7% reduction in life span per 100 rad was chosen. Andersen and Rosenblatt[68] have recently reported the results of a study on female Beagle dogs where the median life span was shortened 6.7% per 100 rads, a value

in complete agreement with our estimate. Storer,[69] however, has stated in a recent publication that 1% per 100 rad is probably a conservative number when extrapolated to exposures occurring at low dose rates.

There has also been a recent analysis[70] indicating that the total increase in all radiogenic cancers (excluding leukemia) might be more than an order of magnitude higher than our estimate. We are not convinced that the data available at the present time justify such a conclusion. However, even if such an interpretation should be correct, our inclusion of non-specific life-shortening (which would include losses due to death by cancer as well as all other causes) in the evaluation as lives lost would be adequate to cover such an effect. Therefore, when our estimate for lives lost due to life-shortening is combined with the estimate for genetic deaths, we feel the total is a conservative upper limit value for the overall risk estimate.

ACKNOWLEDGMENT

This work was performed under the auspices of the U.S. Atomic Energy Commission.

Reference to a company or product name does not imply approval of the product by the University of California or the U.S. Atomic Energy Commission to the exclusion of others that may be suitable.

TABLE 1

Whole Body Dose in Rems for Inhalation and Ingestion of Tritium

	Accident Case		Distance from	Flaring Case		Total Dose for
	Daytime	Nightime	Source	Daytime	Nightime	3 Day Flaring Operation
*† Air Inhalation (12 hrs at highest concentration)	3×10^{-5}	3×10^{-8}	5 km	2×10^{-6}	4×10^{-9}	~ 6×10^{-6}
	7×10^{-6}	2×10^{-6}	15 km	5×10^{-7}	2×10^{-7}	~ 2×10^{-6}
† Dry Deposition (via forage-cow-milk food chain)††	2×10^{-1}	2×10^{-4}	5 km	10^{-2}	10^{-5}	~ 3×10^{-2}
	4×10^{-2}	10^{-2}	15 km	3×10^{-3}	10^{-3}	~ 10^{-2}
† Dry Deposition (via soil-root food chain)**	10^{-2}	10^{-5}	5 km	6×10^{-4}	6×10^{-7}	~ 2×10^{-3}
	3×10^{-3}	7×10^{-4}	15 km	2×10^{-4}	6×10^{-5}	~ 6×10^{-4}
Self-Induced*** Rainout (via forage-cow-milk food chain)††	1.8		2 km	0.36		

* Standard man inhales 2×10^{7} cm^3 per day (ICRP)[4]

† NVO source terms[2,3]

†† Ng et al.[48] dose to a child via forage-cow-milk pathway

** Burton[49], Ng et al.[48] dose to a child via soil-root pathway with modification (see text)

*** Knox, J.[47]

TABLE II

Summary of the Upper Limit Risk to 1000 People Due to the Exposure to 30 mrem in One Year**

	Increased Deaths Due to Radiation	Naturally Occurring Deaths per 1000 People
First Generation		
†Genetic Death	5.4×10^{-3}	200
Adult Leukemia	6×10^{-4}	5
Childhood Leukemia	2×10^{-4}	0.023
Other Adult Malignancy	1.2×10^{-3}	100
Other Childhood Malignancy	3.9×10^{-4}	0.023
*Non-Specific Life Shortening	2.1×10^{-2}	
Total for First Generation (Non-Specific Shortening Plus Genetic Deaths)	$\sim 2.6 \times 10^{-2}$	
Over All Time		
Additional Genetic Deaths	2.2×10^{-1}	
Total Detriment Over All Time	$\sim 2.4 \times 10^{-1}$	

These are upper limit risks; the lower limit risks could be zero.

*Non-specific life shortening includes cancer deaths plus all other diseases and physiological processes leading to death.

**If exposure due to the food chain were reduced by appropriate measures and the primary exposure were by inhalation, then the dose would 0.01 mrem and the above risk factor would be reduced by a factor of 3000.

†Genetic death refers to the eventual elimination of a deleterious gene. This would be evidenced by abortion, stillbirth, pre-reproductive death, early embryoric death, lowered fertility, or sterility.

REFERENCES

1. Project Rulison Definition Plan, CER Geonuclear Corporation Report (1969).
2. Project Rulison Postshot Activities, U.S. Atomic Energy Commission, Nevada Operations Office, Rept. NVO 61 (Preliminary) (November, 1969).
3. Project Rulison Postshot Plans and Evaluations, U.S. Atomic Energy Commission, Nevada Operations Office, Rept. NVO 61 (December, 1969).
4. "Report of ICRP Committee II on Permissible Dose for Internal Radiation (1959)," Health Phys. 3 (1960).
5. Radiation Protection Guidance for Federal Agencies, Federal Radiation Council, Rept. No. 1 (1960).
6. "Standards for Radiation Protection," Chapter 0524, AEC Manual.
7. Evans, A.G., "New Dose Estimates from Chronic Tritium Exposures," Health Phys. 16, 57 (1969).
8. Koranda, J.J., J.R. Martin and R.W. Wikkerink, "Biological Half-Life Studies of Tritium in Animals with a Unique Chronic Exposure at Sedan Crater," Paper presented at the Health Physics Society Annual Meeting, Denver, Colorado, June 16-20, 1968.
9. Thompson, R.C. and J.E. Ballou, "Studies of Metabolic Turnover with Tritium as a Tracer. V. The Predominantly Non-Dynamic State of Body Constituents in the Rat," J. Biol. Chem. 223, 795 (1956).
0. Taylor, R.T., Lawrence Radiation Laboratory, Livermore, Personal Communication.

11. Feinendegen, L. E., Tritium-Labeled Molecules in Biology and Medicine, Academic Press, New York (1967).

12. Siri, W. and J. Evers, "Tritium Exchange in Biological Systems," in Tritium in the Physical and Biological Sciences II, International Atomic Energy Agency, Vienna, 1962, p. 71.

13. Snyder, W.S., B.R. Fish, S.R. Bernard, M.R. Ford and J.R. Muir, "Urinary Excretion of Tritium Following Exposure of Man to HTO - A Two Exponential Model," Phys. Med. Biol. 13, 547 (1968).

14. Reinig, W.C. and S.M. Sanders, Jr., "Assessment of Tritium in Man," in Proceedings of the Symposium on Diagnosis and Treatment of Deposited Radionuclides, CONF-670521-12, Dep. CFSTI, Washington (1967) or as Savannah River Laboratory, Rept. DP-MS-66-77 (1967).

15. "Report on the Meeting of the International Commission on Radiological Protection (ICRP), April, 1969," Acta Radiol. 8, 258 (1969).

16. Butler, H. L. and Leroy, J. H., "Observation of Biological Half-Life of Tritium," Health Phys. 11, 283 (1965).

17. Richmond, C.R., W.L. Langham and T.T. Trujillo, "Comparative Metabolism of Tritiated Water by Mammals," J. Cell Comp. Physiol. 59, 45 (1962).

18. Radford, E.P., T. Merz, C. Ray, R.M. Herriott, C.W. Kruse, C.H. Southwick and B.K. Sladen, "Statement of Concern," Environment 11 (7), 20 (September, 1969).

62

19. Smith, T.E. and R.T. Taylor, Incorporation of Tritium from Tritiated Water into Carbohydrates, Lipids, and Nucleic Acids, Lawrence Radiation Laboratory, Livermore, Rept. UCRL-50781, in press.

20. Metzger, H.P., M. Cuenod, A. Grynbaum and H. Waelsch, "The Use of Tritium Oxide as a Biosynthetic Precursor of Macromolecules in Brain and Liver," J. Neurochem. 14, 99 (1967).

21. Hinrichs, J.R., R.O. Petersen and R. Baserga, "Incorporation of Thymidine into DNA of Mouse Organs," Arch. Pathol. 78, 245 (1964).

22. Rubini, J.R., E.P. Cronkite, V.P. Bond and T.M. Fliedner, "The Metabolism and Fate of Tritiated Thymidine in Man," J. Clin. Invest. 39, 909 (1960).

23. Lambert, B.E. and R.J. Clifton, "Radiation Doses Resulting from the Ingestion of Tritiated Thymidine by the Rat," Health Phys. 15, 3 (1968).

24. Guild, W.R., "Hazards from Isotopic Tracers," Science 128, 1308 (1958).

25. Hatch, F.T., J.A. Mazrimas, G.G. Greenway, J.J. Koranda and J.L. Moore, Studies on Liver DNA in Tritiated Kangaroo Rats Living at Sedan Crater, Lawrence Radiation Laboratory, Livermore, Rept. UCRL-50461 (1968).

26. Moore, R., "A Comparison of HTO in Plasma and Expired Water Vapor," Health Phys. 7, 161 (1962).

27. Bowen, J. H. M., Trace Elements in Biochemistry, Academic Press, New York (1966).

28. Bond, V. P. and L. E. Feinendegen, "Intranuclear ^{3}H Thymidine: Dosimetric, Radiobiological and Radiation Protection Aspects," Health Phys. 12, 1007 (1966).

29. Marin, G. and M. A. Bender, "A Comparison of Mammalian Cell-Killing by Incorporated ^{3}H-Thymidine and ^{3}H-Uridine," Intern. J. Radiation Biol. 7, 235 (1963).

30. Marin, G. and M. A. Bender, "Survival Kinetics of HeLa S-3 Cells after Incorporation of ^{3}H-Thymidine or ^{3}H-Uridine," Intern. J. Radiation Biol. 7, 221 (1963).

31. Drew, R. M. and R. B. Painter, "Action of Tritiated Thymidine on the Clonal Growth of Mammalian Cells," Radiation Res. 11, 535 (1959).

32. Drew, R. M. and R. B. Painter, "Further Studies on the Clonal Growth of HeLa S-3 Cells Treated with Tritiated Thymidine," Radiation Res. 16, 303 (1962).

33. Painter, R. B., R. M. Drew and B. G. Giaugue, "Further Studies on Deoxyribonucleic Acid Metabolism in Mammalian Cell Cultures," Exptl. Cell Res. 21, 98 (1960).

34. Lambert, B. E., "Cytological Damage Produced in the Mouse Testes by Tritiated Thymidine, Tritiated Water and X-Rays," Health Phys. 17, 547 (1969).

35. Johnson, H.A. and E.P. Cronkite, "The Effect of Tritiated Thymidine on Mouse Spermatogonia," Radiation Res. 11, 825 (1959).

36. Dewey, W.C., R.M. Humphrey and B.A. Jones, "Comparisons of Tritiated Thymidine, Tritiated Water and Cobalt-60 Gamma Rays in Inducing Chromosomal Abberations," Radiation Res. 24, 214 (1965).

37. Bender, M.A., P.C. Gooch and D.M. Prescott, "Aberrations Induced in Human Luekocyte Chromosomes by ^{3}H-Labeled Nucleosides," Cytogenetics 1, 65 (1962).

38. Baserga, R., H. Lisco and W.E. Kisieleski, "Tumor Induction in Mice by Radioactive Thymidine," Radiation Res. 29, 583 (1966).

39. Bateman, A.J. and A.C. Chandley, "Mutations Induced in the Mouse with Tritiated Thymidine," Nature 193, 705 (1962).

40. Greulich, R.C., "Deleterious Influence of Orally Administered Tritiated Thymidine on Reproductive Capacity of Mice," Radiation Res. 14, 83 (1961).

41. Kaplan, W.D., H.D. Gugler, K.K. Kidd and V.E. Tinderholt, "Nonrandom Distribution of Lethals Induced by Tritiated Thymidine in Drosophila Melanogaster," Genetics 49, 701 (1964).

42. Kaplan, W.D., H.D. Gugler and K.K. Kidd, "Distribution of Lethals Induced by Tritiated DNA Precursors in Drosophila Melanogaster," Genetics 53, 499 (1966).

43. Rachmeler, M. and A.B. Pardee, "Loss of Viability and β-Galactosidase-Forming Ability as a Consequence of Tritium Decay in Escherichia coli," Biochim. Biophys. Acta 68, 62 (1963).

a65

44. Khan, A.A. and J.E. Wilson, "Studies of Turnover in Mammalian Subcellular Particles: Brain Nuclei, Mitochondria and Microsomes," J. Neurochem. 12, 81 (1965).

45. International Commission on Radiological Protection, The Evaluation of Risks from Radiation, ICRP Publication 8, Pergamon Press, New York (1966).

46. Miller, R.W., "Delayed Radiation Effects in Atomic-Bomb Survivors," Science 166, 569 (1969).

47. Knox, J.B., Meterological Aspects of the Rulison Reentry, Lawrence Radiation Laboratory, Livermore, Informal Rept. UOPKA 69-54 (1969).

48. Ng, Y.C., C.A. Burton, S.E. Thompson, R.K. Tandy, H.K. Kretner and M.W. Pratt, Prediction of the Maximum Dosage to Man from the Fallout of Nuclear Devices. IV. Handbook for Estimating the Maximum Internal Dose from Radionuclides Released to the Biosphere, Lawrence Radiation Laboratory, Livermore, Rept. UCRL-50163 Part IV (1968).

49. Burton, C.A., A Graphical Method for Calculating the Dose in Man from Individual Radionuclides in Fallout, Lawrence Radiation Laboratory, Livermore, Rept. UCRL-14922 (1966).

50. Koranda, J.J. and J.R. Martin, "The Persistence of Radionuclides at Sites of Nuclear Detonations," in Biological Implications of the Nuclear

#66

Age, Proceedings of the Bio-Medical Symposium held at the Lawrence Radiation Laboratory, Livermore, March 5-7, 1969, CONF-690303, p. 159 (1969).

51. Martin, J.R., C.F. Jordan, J.J. Koranda and J.R. Kline, "Radioecological Studies of Tritium Movement in a Tropical Rain Forest," in Proceedings of a Symposium on Engineering with Nuclear Explosives, January 14-16, 1970, Las Vegas, Nevada (to be published).

52. Pinson, E. and W.H. Langham, "Physiology and Toxicology of Tritium in Man," J. App. Physiol. 10, 108 (1957).

53. Russell, W.L., "The Effect of Radiation Dose Rate and Fractionation of Mutation in Mice," in Repair from Genetic Radiation Damage, F.H. Sobels, Ed., Oxford: Pergamon Press (1963).

54. Russell, W.L., "Recent Studies on the Genetic Effects of Radiation in Mice," Pediatrics 41, 223 (1968).

55. Elkind, M.M., C. Kamper, W.B. Moses and H. Sutton-Gilbert, "Sublethal-lethal Radiation Damage and Repair in Mammalian Cells," Recovery and Repair Mechanisms in Radiobiology, Brookhaven National Laboratory Symposium, Rept. BNL-50058, p. 134 (1967).

56. Wolff, S., "Some Postirradiation Phenomena that Affect the Induction of Chromosome Abberations," J. Cell. Comp. Physiol. (Supp. 1) 151 (1961).

57. Wolff, S., "On the Chemistry of Chromosome Continuity," Natl. Cancer Inst. Monogr. 18, 155 (1965).

58. Cleaver, J.E. and R.B. Painter, "Evidence for Repair Replication of HeLa Cell DNA Damaged by Ultraviolet Light," Biochim. Biophys. Acta 161, 552 (1968).

59. Rasmussen, R.E. and R.B. Painter, "Radiation-Stimulated DNA Synthesis in Cultured Mammalian Cells," J. Cell Biol. 29, 11 (1966).

60. Cleaver, J.E., "Repair Replication of Mammalian Cell DNA: Effects of Compounds that Inhibit DNA Synthesis in Dark Repair," Radiation Res. 37, 334 (1969).

61. Painter, R.B. and J.E. Cleaver, "Repair Replication, Unscheduled DNA Synthesis, and the Repair of Mammalian DNA," Radiation Res. 37, 451 (1969).

62. Painter, R.B. and J.E. Cleaver, "Repair Replication in HeLa Cells after Large Doses of X-irradiation," Nature 216, 369 (1967).

63. Lett, J.T., I. Caldwell, C.J. Dean and P. Alexander, "Rejoining of X-Ray Induced Breaks in the DNA of Leukemia Cells," Nature 214, 790 (1967).

64. Weiss, B. and C.C. Richardson, "Enzymatic Breakage and Joining of Deoxyribonucleic Acid, I. Repair of Single-Strand Breaks in DNA by an Enzyme System from Escherichia coli Infected with T4 Bacteriophage," Proc. Natl. Acad. Sci. U.S. 57, 1021 (1967).

65. Zimmerman, S.B., J.W. Little, C.K. Oshinsky and M. Gellert, "Enzymatic Joining of DNA Strands: A Novel Reaction of Diphosphopyridine Nucleotide," Proc. Natl. Acad. Sci. U.S. 57 1841 (1967).

66. Kelly, R.B., M.R. Atkinson, J.A. Huberman and A. Kornberg, "Excision of Thymine Dimers and Other Mismatched Sequences by DNA Polymerase of Escherichia coli," Nature 224, 495 (1969).

67. Anspaugh, L.R. and W.L. Robison, Quantitative Evaluation of the Biological Hazards of Radiation Associated with Project Ketch, Lawrence Radiation Laboratory, Livermore, Rept. UCID-15325 (1968).

68. Andersen, A.C. and L.S. Rosenblatt, "The Effect of Whole-Body X-Irradiation on the Median Lifespan of Female Dogs (Beagles)," Radiation Res. 39, 177 (1969).

69. Storer, J.B., "Late Effects: Extrapolation to Low Dose Rate Exposures," Health Phys. 17, 3 (1969).

70. Gofman, J.W. and A.R. Tamplin, "Low Dose Radiation, Chromosomes, and Cancer," Proc. of the IEEE 16th Nuclear Science Symposium, in press.